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DIAGNOSIS AND CLINICAL MANIFESTATIONS OF CARDIO-SPASM ASSOCIATED WITH DIFFUSE DILATATION OF THE ESOPHAGUS.[1]

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I. **General Consideration.** Definition. A condition in which there occur diffuse dilatation of the esophagus with maintenance of the normal contour of the gullet and a varying degree of unusually strong and prolonged contraction or hypertrophy of the cardiac sphincter.

There may coexist hypertrophy of the muscle layers of the lower esophageal wall. The hypertrophy of the cardiac sphincter may be of but moderate extent, but when it is associated with powerful spasmodic or long-continued contraction there results transient or long-maintained, frequently complete obstruction to the passage of esophageal contents from the gullet into the stomach. Again, the hypertrophy of the cardiac sphincter may be advanced and then such hypertrophy may of itself result in functional stenosis of the cardia, whether or not there is the complication of associated spasm. In marked instances of the affection, the hypertrophy of the cardiac sphincter may resemble in degree such hypertrophy as is found at the pyloric sphincter in patients affected with the hyper-

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trophic stenosis of infancy. Cases of "cardiospasm" described in which muscular hypertrophy of the cardiac sphincter could not be demonstrated at autopsy or operation would seem to be errors in diagnosis or faults in the observation of what actually constitutes muscular hypertrophy in a dilated, enlarged, static esophagus. There have been too few studies of the normal cardiac sphincter to permit comparison. During the past few years many papers have appeared purporting to describe "cardiospasm," but, in truth, such have dealt with instances of spasm at the cardia or of transient esophageal spasm, local or general.

NOMENCLATURE. It should be understood definitely that the terms "spasm at the cardia" and "cardiospasm" are not synonymous. Only the latter form of affection includes hypertrophy of the cardiac sphincter or the wall of the lower portion of the esophagus, combined with general, diffuse dilatation of the esophagus. "Spasm at the cardia" is a transient lesion of itself. It is not associated with uniform, diffuse dilatation of the esophagus until it has been so long existent or is of such constant maintenance as to produce esophageal changes not of a transient nature, particularly an associated, permanent dilatation of the gullet.

ETIOLOGY. In cardiospasm the dilatation of the esophagus would appear to be due to rather indefinitely understood causes. Rosenheim has suggested, as an explanation, primary atony of the muscular coats of the gullet. Kraus considers the condition due to the development of persistent, long-maintained spasm of the cardia, associated with paralysis of the circular muscles of the esophagus, the latter occurring as a consequence of degenerative changes in the vagi. Numerous authors, as, for example, Fleiner, Zenker and Sievers, maintain that dilatation results from a congenital predisposition on the part of the esophagus. Martin considers a primary esophagitis as an important factor. In addition there have been advanced other explanations, viz.: kinking of the esophagus at the cardiac opening; anatomical defects associated with the esophagus or the stomach, such as ulcer or carcinoma, and congenital or acquired asthenia. Plummer, following Mikulicz and Meltzer, is inclined to the opinion that, in addition to the changes occurring at the cardiac sphincter, there exists some disturbance of the nerve-muscle mechanism of the esophagus which may permit diffuse dilatation of the gullet irrespective of actual stenosis due to hypertrophy of the cardiac sphincter. Plummer emphasizes that in some patients there may be noted the occurrence of a habit of spasm at the cardia, and this, without anatomical, diffuse dilatation of the esophagus. Numerous such instances have found their way into the literature, misnamed "cardiospasm."

In our opinion it is likely that such extra-esophageal lesions as give rise to repeated, powerful, long-maintained contractions of the cardia are capable of producing the muscular hypertrophy of over-

work; coincidently, this alteration in sphincter reflex-time secondarily disturbs the normal neuromuscular coördination in the esophagus above it. As a result, the overworked sphincter seems to develop its own independent contraction rate, and this of a duration beyond the normal time limits and out of the normal rhythm of the swallowing act. In such circumstances, the stimuli from above (formerly passing down to a cardiac sphincter, normally contracting and relaxing in appropriate time and with limited degree) are delivered in an uncoördinated fashion and at irregular time intervals, with little or no effect upon the systole and diastole of the cardiac sphincter. The initial overstimulation to the cardia would seem to be exerted by numerous anomalies. Such may be extra-esophageal or intrinsically esophageal. These stimuli may arise from the central nervous system, from the esophagus itself, from the diaphragm, the intrathoracic organs, from the stomach, duodenum, gall-bladder, appendix or other abdominal viscera. In some patients it would seem that more than usually powerful cardiac contractions are initiated by psychic upsets. However, it should be emphasized that cardiospasm is by no means an affection only of psychasthenics or of neurologically unstable individuals. While the initial cause of spasm at the cardia may produce a more active response in individuals who are psychically or nervously abnormally responsive to all stimuli, it should be recognized that in well-developed instances of cardiospasm the lesion which has to be dealt with is an anatomical one and not a mere functional disturbance. In these patients, in spite of the opinion to the contrary held by some clinicians, I believe there always exists a definite hypertrophy of the muscular cardiac ring (such as can be shown when skilled fluoroscopic study is made during the taking of food). With this is always readily demonstrable (although of varying degree) generalized dilatation of the esophagus. It is important that these facts be recognized because upon their realization depends the exhibition of proper methods of treatment. Patients affected with this lesion require more accurate therapeutic procedures than rest-cures, cessation from mental, psychic or physical overactivity, quieting medicines, Christian Science, osteopathic hocus-pocus and the like.

Structural Changes in the Esophagus. The anatomical anomalies present in cardiospasm should be emphasized, viz.: hypertrophy of the cardiac sphincter, atypic cardiac contractures (powerfully spasmodic or abnormally persistent) and diffuse enlargement of the esophagus without local or general gross alterations of its contour. The effect of these anatomical changes is to cause dysphagia, imperfect passage of foods into the stomach and, generally, some degree of permanent esophageal retention. The quantity of retained food varies widely in different patients and in a given patient at various times during the study of his affection. The

esophagus may retain from less than an ounce to as much as several liters of fluid or mixed food. Upon the patient, the systemic effects are those consequent upon slow or rapid starvation. The degree of malnutrition may be so pronounced and of such acute development as to suggest malignant disease or advanced cicatricial stenosis caused by peptic, luetic, tuberculous or traumatic ulcer of the gullet.

II. Material Studied. During the past seven years 76 instances of cardiospasm associated with diffuse dilatation of the esophagus have come under my observation. Tabulated records of 47 of these cases are available for study.

Sex. Of the 47 cases there were 25 females and 22 males. This approximate equality of the sex ratio is rather striking in view of the prevalent opinion among practitioners that females are much more likely to be affected than are males.

Age. The average age for the series was 39.2 years; the youngest case was aged nineteen and the oldest seventy years. It is thus seen that true cardiospasm is a disease common to midadult life, the majority of instances occurring between twenty-five and thirty-five years.

Occupation. Occupation appears to play a very small part as an etiological factor, but the ailment seems to be relatively common in those individuals who are very active, physically and mentally. Temperament has been rather unduly emphasized in its relation to the initiation of cardiospasm. While it is true that some of these patients are delicately balanced psychically, yet a goodly number are of what one would call stable temperament.

Ailments previous to the onset of cardiospasm, with a few exceptions, appear to have little bearing with regard to causing the affection. It was noticed in our series that in 8 instances frequently recurring attacks of bronchitis or asthma were associated with dyspepsia and ultimately with dysphagia. These severe coughing spells might be a not unimportant factor in causing cardiac irritation and neuromuscular fatigue of the diaphragm. There were 3 instances of gastric ulcer proved by laparotomy, but the cardiospasm was in no way influenced by the operative procedure upon the stomach. In but 7 instances did shock, fright or acute fatigue precipitate the symptoms (2 were neuroses). Excessive smoking, particularly of cigarettes, was observed in 5 cases. This might offer an interesting etiological suggestion to those who attribute cardiospasm to vagus malfunction.

Mode of Onset. In but 17 instances the affection with the associated dysphagia was acute in its inception (36 per cent.). In 30 cases (74 per cent.) the affection appeared gradually and was not infrequently punctuated by attacks of exaggerated distress.

Duration of All Symptoms. This averaged for the series 5.6 years and ranged in the individual cases from three months to twenty years. The degree of dysphagia or esophageal retention not infre-

quently bore no relation whatever to the time that symptoms had been present. Not rarely, esophageal retention of as great as 1000 c.c. arose in individuals who had been ill for less than a year (9 cases).

The duration of the presenting trouble averaged for the series 4.2 years, with a range in the individual cases of from two months to twenty years.

DYSPHAGIA is not commonly painful. Usually there is a sensation of fulness or tension beneath the sternum, a feeling of tightness or a discomfort often described as "crowding of the heart." There may be dyspnea or cough. The first swallows of food may produce the uncomfortable sensation. It is of diagnostic significance to know that liquid foods are more prone immediately to bring about distress than are solids. In this way the type of dysphagia differs strikingly from that common to various forms of anatomical stenosis (ulcer, cancer, trauma, etc.) along the course of the esophagus. There are instances, however, in which the swallowing-act at once excites sharp cramp-like pains, usually located beneath the xiphoid or along the spine, from the ninth to the twelfth dorsal vertebræ. The pain may be at first sufficiently acute to cause incapacitation and then may be followed by intermittent or continuous gripping, binding or a "sticking" type of discomfort.

In our series of patients there was *constant* dysphagia in 45 cases. There was dysphagia to *fluids only* in 21, to *solids only* in 14 and to *all foods* in 12 cases.

Dysphagia is nearly always accompanied by *vomiting* or spasmodic food regurgitation so long as esophageal muscle tone remains good. A fairly characteristic and differential feature of the vomiting is that it is usually sudden, frequently explosive and very often occurs shortly after the ingestion of food. Liquids are more liable to produce sudden, copious emesis than are solids; in fact, the ingestion of soup or fluid at the early part of a meal may bring about spasmodic emesis of such sudden and forceful nature that the individual is compelled to rush hastily from the table to avoid accidents. This prompt vomiting of liquids is frequently aggravated by the fact that, but rarely, in general practice is the lesion of cardiospasm recognized. To the average physician a dysphagia presumably indicates a liquid diet, and he accordingly orders such. Solids or partly solid foods are generally well borne, particularly in the early stages of cardiospasm. The constant attempt to take liquids is followed promptly by vomiting, lack of nourishment and malnutrition, and doubtless added damage to the weakened esophageal wall. The patient frequently finds this out himself and refuses to follow his physician's instructions when liquid diet has been urged. In advanced cardiospasm practically all the food taken may be vomited within an hour or two following its ingestion, or if dilatation of the esophagus is pronounced, morning retention-vomiting is common and constant.

The vomitus consists commonly of material whose appearance resembles that of food as eaten. It is alkaline in reaction, usually contains much mucus, rarely lactic acid or blood and may have a high, bacterial content. At times vomiting is "delayed" and only when a large meal is taken at night is emesis produced. At that time, food eaten at breakfast or even the night before may be vomited unchanged. Sometimes the vomitus is copious at night and prevents proper rest and sleep. A few cases of even marked cardiospasm do not manifest actual vomiting, but there is constant belching, bad taste in the mouth, eructations of food and, frequently, copious regurgitation of ill-tasting fluid, food or mucus. Sometimes nausea is pronounced and there occurs remarkable increase in the secretion of saliva. This may indicate central irritation of the salivary glands. Even when some food passes the cardia the regurgitation of frothy, thick, tenacious mucoid material is apt to be annoying, particularly when such occurs toward evening. Consequent upon the vomiting appear evidences of lack of fluid in the tissues, constipation, diminution of urine output, headaches, dizziness and anemia.

In this series of cardiospasm patients, 31 vomited daily (frequently many times during the day). In 22 cases retention-vomiting was observed. Frequently, food (fruit skins, pieces of tomato, meat, lettuce, milk-clots, etc.) was retained in the lower part of the dilated esophagus for as long as a week. In 17 of our patients the vomiting was projectile. In such cases it was a common experience that patients began vomiting as soon as food was swallowed. There was vomiting of blood in 14 cases. This hematemesis, seemingly, bore relationship to dilated or varicose veins in the lower esophagus, although one could not always be certain that ulceration had not taken place in some portion of the esophageal wall where varicosities were not present. In one case the hematemesis appeared to be definitely "vicarious," inasmuch as it occurred on two occasions just before menstruation.

Regurgitation, without actual vomiting, was especially annoying in fifteen patients. This regurgitation was generally uncontrollable and the regurgitant food-mass was frequently very copious in quantity and of a foul, sickening odor. There were patients in which periods of intermittent regurgitation preceded cycles of exhausting vomiting.

Miscellaneous Clinical Symptoms. Weight loss was experienced by all our patients. It averaged, for the series, 28.2 pounds. In individual cases it ranged from ten to as much as eighty-two pounds. The appetite was stated as being lessened or as being poor in 44; 9.5 per cent. patients experienced pronounced anorexia. Normal bowel movements occurred in 47 per cent. In the remaining cases there was obstinate constipation. This constipation seemed to be due largely to insufficient quantities of food or fluid passing the cardia into the stomach and bowel.

III. Special Diagnostic Maneuvers.—In true cardiospasm, an unguided *stomach tube* passed into the esophagus usually excites free food regurgitation about the tube and the spasmodic emesis of much food and liquid through the tube. The tube may be generally passed freely as far as the normal cardia, but there it meets an elastic resistance. This resistance is occasioned either by the tube's impinging on the distended esophageal wall and depressing its lower sixth into a sac-like pouch, or by pronounced and persistent contracture of the cardiac sphincter. In only but a few not well-established instances of the affection does the cardia lie in a line direct from the pharynx and thus allow an unguided stomach tube to be passed to it, and then, upon exertion of pressure, admit of the cardia's being forced, the tube being carried into the stomach. If the tube is guided upon a silk cord to the cardia (as suggested by Mixter, of Boston, seventeen years ago and emphasized by Henry Plummer clinically), it can be carried direct to the cardia, and, provided the tube is a stiff one, can, with the exertion of a moderate degree of force, be passed into the stomach. The "giving way" of the tight, elastic cardia can be felt as the stomach tube passes the hypertrophied or permanently contracted sphincter.

If not guided to the cardiac orifice by a cord, a *bougie* passed into the esophagus traverses the lumen farther than the average distance of the cardia from the incisor teeth. Usually the olive may be passed considerably farther than normally and the unsuspecting manipulator may fancy that he is well beyond the cardia and that the olive lies in the stomach, it having passed the cardiac sphincter without the operator's knowledge. This mistake occurs as a result of the bougie's passing downward and to the right in the folds of the sac-like, pendulous lower esophagus. In such circumstances forcible pressure is dangerous: in our own experience one case of esophageal rupture occurred after the patient had been elsewhere "dilated by olives" for six months and the cardia was later divulsively dilated. At the autopsy it was found that at the lowest point of the dilated esophageal sac, and about two inches from the cardia, there were denudation of the mucous membrane, marked thinning of the wall of the esophagus and peri-esophageal inflammatory thickening. The area of the esophageal attenuation was about the size of a large olive, somewhat depressed, and lay in a straight line from the pharynx to the lowest part of the esophageal sac, but was out of the straight line from pharynx to cardia. In this patient, a divulsing dilator, guided upon a silk cord, passed freely into the cardiac orifice, but the increase in intra-esophageal pressure during dilatation was sufficient to cause rupture of the esophagus at the spot where repeated bouginage had weakened the walls.

Even in long-established, obstinate cardiospasm it is generally possible for a silk cord guide to pass from the esophagus into the stomach, but at times the cord may be held in the esophagus several

days and may pass freely only when spasmodic contracture of the cardia has been minimized by the free use of atropin or of belladonna. However, unless the dilated esophagus be washed clean of retention contents silk cords may lie for weeks without passing from the gullet to the stomach.

An olive bougie of large size passes readily to the cardia upon a taut string; moderate pressure "forces" the sphincter and permits the bougie's passing into the stomach. Withdrawal of the bougie may be accompanied by a readily recognizable, elastic resistance. It is usually the experience that the bougie exerts little influence with respect to permanent enlargement of the cardiac opening or permanent relief of the dysphagia. It is quite as sensible to expect permanent dilatation of a cardia in cardiospasm by bouginage as it is to expect that a spasm of the anal sphincter will be relieved by frequent digital examinations. If the patient be examined at different times of the day or upon different days a variation in the amount of resistance at the cardia may be determined, but it is characteristic of the affection that at every examination there is resistance present.

The roentgen examination is of great aid in diagnosis but the evidence which it supplies is not infallible. Fluoroscopic observation and plate studies must be carefully interpreted in the light of clinical history, physical examination and examination of the esophagus by guided instruments.

Fluoroscopically when the patient swallows the opaque meal it will be seen that usually a small amount of the mixture immediately passes through the cardia into the stomach. In moderately well-established instances of the affection the cardia then closes quickly, with or without several, easily recognized, squirty contractions and continued ingestion of the opaque mixture results only in filling out the esophagus; practically nothing passes into the stomach. It can then be shown that the esophagus is uniformly dilated, that its wall is regular, that dilatation occurs along anatomical lines and that the cardiac end of the esophagus down to the hypertrophied sphincter presents no gross irregularities of contour. In shape such a dilated esophagus resembles, characteristically, a large lily-of-the-valley leaf, or is roughly pear-shaped. The dilatation may be enormous and extend so markedly throughout the entire length of the esophagus as to produce pressure upon the heart, the aorta or the trachea. The obstruction at the cardia may be absolute and may be maintained for hours or days until emesis or lavage results in an apparently reflex partial relaxation of the hypertrophied cardiac sphincter. Again, in moderately advanced cases esophageal retention may persist from a half to two hours and then the opaque meal passes in a jerky, spasmodic fashion, slowly through to the stomach. Even in these cases, however, one rarely finds that the esophagus completely empties itself within from two to six hours. In the early, incompletely established cases esophageal retention may be but a tran-

sient one. Fluoroscopically it is possible to observe alternate, spasmodic contractions and relaxations of the cardiac sphincter,

FIG. 1.—Lateral roentgenogram, showing bismuth meal retention in case of "cardiospasm." *A*, closed cardia, without malformation; *B*, diffusely dilated esophagus, normal contour maintained; *C*, anterior depression caused by aortic arch; *D*, shadow of heart. (Roentgenogram by Joseph Johnson.)

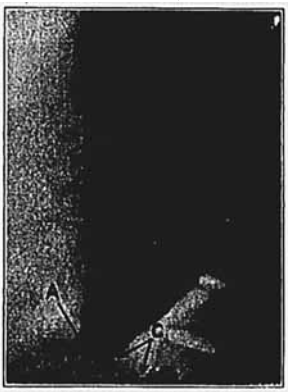

FIG. 2.—Lateral roentgenogram of bismuth meal retention in case of "cardiospasm." *A*, cardia, in contraction, without malformation, but with stenosis complete; *B*, diffusely dilated esophagus of rather marked degree, but contour normal; *C*, diaphragm line. (Roentgenogram by Joseph Johnson.)

associated with the intermittent passage of small amounts of the opaque meal from the lower esophagus into the stomach. The esophagus will thus empty in from a few minutes to an hour. Mild

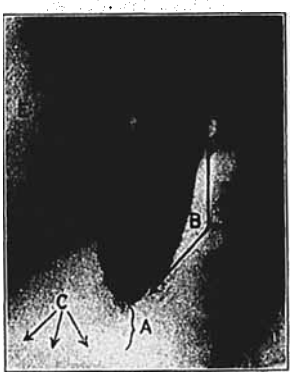

FIG. 3.—Lateral roentgenogram, showing retention of bismuth meal in a case of ring cancer involving the lower one-sixth of the esophagus, with complete stenosis. *A*, zone of cancerous involvement; *B*, dilated esophagus, proximal to cancer; *C*, diaphragm line; *D*, heart shadow; *E*, aortic arch. The lower portion of the esophagus has a contour resembling normal, and plate might readily be mistaken for one of "cardiospasm" if care is not exercised in locating the cardiac end of the esophagus in relation to the position of the diaphragm line and the gas shadow in the stomach. (Roentgenogram by Joseph Johnson.)

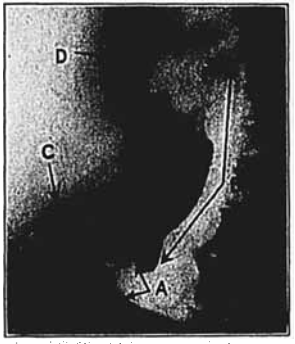

FIG. 4.—Lateral roentgenogram of retained bismuth meal in the esophagus, the cardiac end of which is stenosed by cancer of the nodular, cauliflower type. *A*, zone of cancer, irregularly canalized; *B*, dilated and partly filled esophagus; *C*, heart shadow; *D*, shadow of aortic arch; *E*, dilated, but not filled, esophagus, the dilatation extending above the tracheal bifurcation. (Roentgenogram by Joseph Johnson.)

instances of the affection are not generally associated with great sac-like esophageal dilatation.

In rare instances, fluoroscopy and roentgen plates fail to make the diagnosis, the roentgen findings seem characteristic for cardiospasm, but in the dilatation of the esophageal sac, ulceration, diffuse scirrhus carcinoma or lues may remain masked even until they have advanced sufficiently to produce extensive stricture or to cause death from metastasis. Not rarely, fluoroscopy clearly demonstrates torsion of the terminal esophagus with or without rather sharp angulation or displacement, usually to the left of the midline of the thorax.

In several instances of well-developed cardiospasm with diffuse dilatation of the esophagus I have noted unusual "doming" of the diaphragm. In these patients the diaphragm movements were atypic: degree of depression was unequal on the two sides, rates of downward movements were not equal on the two sides, *i. e.*, the diaphragm depressions were not synchronous and, locally, in the region of the hiatus there seemed to be a sort of "piling up" of diaphragm (local hypertrophy). Observation of the above phenomena has suggested to me that at times the fault primary in initiating cardiospasm lies in the muscle or serous layers of the diaphragm (inflammatory processes in the serous coverings of the esophagus, lungs, heart, diaphragm or a local myositis of the diaphragm itself), with resultant disturbance in the neuromuscular mechanism of the diaphragm, and, subsequently, of adjacent viscera with their correlated nerve supply.

It should be scarcely necessary to mention that fluoroscopy, conducted with the patient only in the anteroposterior position, can at its best give but incomplete information. Unless the patient is examined also laterally so that the course of and the anomalies in the esophagus are susceptible of complete exposure much of interest and of clinical usefulness will be lost. It is advisable to examine fluoroscopically during the swallowing act with the diaphragm depressed by deep inspiration, the breath being held and the diaphragm fixed during swallowing. The patient may be turned rapidly from the anteroposterior to the lateral ("quartering") position, and by this maneuver one may secure views of the lowest portion of the esophagus, of the sphincter, the contour, peristaltic activity and the position and shape of the diaphragm. For careful scrutiny of the terminal esophagus and the cardiac sphincter the fluoroscope's diaphragm should be closed to such point as will permit minute study of the esophageal contour inch by inch. Such procedure is necessary to detect or rule out ulcers, scirrhus carcinoma, etc., and to properly study the sphincter and its action. The successive employment of thin fluid and thick mush-like, opaque suspensions gives useful information relative to sphincter action, esophageal peristalsis, stasis, contour and degree of dilatation.

The patient should be examined at least on two successive days. The second study should be carried on after the exhibition of antispasmodic medicines to physiological tolerance. Only in this way can one determine the true condition existing at the cardia and the actual size of the esophagus. If this procedure were routinely followed, many "literature" cardiospasms would disappear—for many such are only "spasm at the cardia."

Roentgenograms should be made with the patient in the semilateral or "quartering" position as well as the anteroposterior. When plates are made with the patient in the semilateral position the entire esophageal contour may be delimited. In order to bring out the cardia and the lower portion of the esophagus it is advisable to have the patient drink as much of the opaque meal as he is able to, then clasp his hands high above his head and take a deep breath. The inspiration depresses the diaphragm and generally enables accurate visualization of the lower esophagus. The best roentgenograms are made with the patient standing with his left side next to the plate. When extensive esophageal dilatation is present, anteroposterior roentgenograms also should be made in order to determine the lateral limit of the esophageal sac and to demonstrate the degree of encroachment of the enlarged esophagus upon the heart, aorta and lungs. Such plates frequently demonstrate that the lowest limit of the dilated esophagus lies several inches below the level of the cardiac sphincter. This shows, vividly, how futile is the effort to pass bougies or tubes into the stomach without their being guided from pharynx to cardia by a swallowed silk thread. In patients in whom the cardiospasm is not far advanced, anteroposterior roentgenograms even may fail to indicate that the condition is present. In such circumstances the esophageal shadow is overlapped and hidden by the shadows of the heart, the aorta and the spine. Series of plates should be made before and after the administration of antispasmodic medicines (belladonna, benzylbenzoate, etc.). Such permit of interesting comparative studies and furnish valuable records of anatomical changes as the functions of the esophagus are restored by treatment.

Esophagoscopy. Inasmuch as in certain cases of cardiospasm the initiating fault lies in an esophageal lesion it would appear a proper procedure to explore by sight the entire esophageal mucosa from pharynx to cardia, that is, provided that one who is familiar with esophagoscopes and who knows the significance of what he sees is able to do the work. Much esophagoscopic work as done is worthless because the operator knows little of the appearance of the normal esophagus. In well-established instances of cardiospasm an actual view of the interior of an enormously dilated gullet and an anomalously acting sphincter is truly worth while if for no other reason than to satisfy one's curiosity. When some doubt exists respecting whether or no a cardiospasm is complicated by

scirrhus cancer, peptic ulcer, foreign body, gumma or extra-esophageal ailment, certainly, esophagoscopy is a justifiable procedure to clear up doubtful points in diagnosis even though it is annoying to the subject. A thoroughly cleansed esophagus, carefully administered intratracheal anesthesia and a good esophagoscope, such as the particularly ingenious Sussmann, greatly facilitate the procedure. When powerful contractures of the cardiac sphincter persistently recur, following most expert divulsive treatment, it would seem necessary always to perform esophagoscopy with the object of searching for and treating locally irritative mucosal lesions.

Prognosis. If the affection is properly managed, there is clinical and functional recovery in about 70 per cent. of even well-established instances of cardiospasm. Improvement occurs in 20 per cent. About 10 per cent. of the patients are not permanently benefited by any form of treatment at the most expert hands. The lesion seemingly resists all forms of therapy. In this class are patients extremely emaciated, instances of irritable esophagus where proper treatment cannot be carried out and very likely cases in which there is a congenital defect, or, as Plummer has emphasized, the persistence of a "habit of spasm." For these unfortunates, gastrostomy may be required to save or prolong life. If with cardiospasm there are such associated lesions as gastric ulcer or malignancy, gall-stones, appendix infection or ulceration or inflammation of the cardia or esophagus itself prognosis is seriously limited unless these lesions are amenable to appropriate medical or surgical treatment. Even after the restoration of the cardiac sphincter action, the esophagus may remain much dilated and permit food retention. However, it is surprising how dilatation of the esophagus may persist, and yet such is unaccompanied by troublesome dysphagia, provided the cardiac orifice is patent and the sphincter acts normally.

THE SPLEEN AND DIGESTION:

Study I.—The Spleen and Gastric Secretion.[1]

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The exact rôle of the spleen in digestion is not known. Numerous hypotheses have been advanced and much experimental work has

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